3:00

WHAT ARE THE PREDICTORS OF ACUTE COMPLICATIONS FOLLOWING CORONARY ARTERY STENTING? SINGLE INSTITUTIONAL EXPERIENCE

Gary S. Roubin, Subodh Agrawal, Larry S. Dean, William A. Baxley, Paul Garrahy, Ravi K. Bajaj, Philip Gainey, J. Bradley Cavender, Peter Macander, Adam Cannon. University of Alabama, Birmingham, Alabama

Predictors of acute complications following coronary stenting with the balloon expandable flexible coil stent (Cook, Inc.) have not been characterized. We analyzed results in 96 consecutive pts from our institution. Deployment success rate was 98% (96/98 pts), 62 underwent acute coronary stenting post percutaneous transluminal coronary angioplasty (PTCA) for acute coronary dissection and 34 underwent elective stenting for restenosis. Elective stenting was associated with fewer complications than acute stenting (stent thrombosis 0 [0%] versus 8 [12%] p = .07, emergent re-PTCA 0 versus 9 [14%] p < .001, emergency CABG 0 versus 2 [3%], p = ns). Of 8 pts who had stent thrombosis, 7 received enteric coated aspirin, 4/8 (50%) had recent myocardial infarction, 5/8 (62%) had residual dissection after stenting, 2/8 (25%) had inadequate anticoagulation. There was a marked institutional learning curve in managing anticoagulation. For the first ½ of the series compared to the second ½, bleeding complications were 17/48 (38%) versus 6/48 (12%), p = 0.02, vascular repair 6/48 (13%) versus 3/48 (6%).

In conclusion for this stent prototype, stent thrombosis remains a significant problem after stenting for acute dissection but not afteer elective use. Bleeding complications can be reduced to acceptable rates with increasing institutional experience.

3:15

STENTING OF VENOUS BYPASS GRAFTS: A NEW TREATMENT MODALITY FOR PATIENTS WHO ARE POOR CANDIDATES FOR REINTERVENTION

Ivan K. De Scheerder, Bradley H. Strauss, Harry Suryapranata, Pim J. de Feyter, Patrick W. Serruys. Thoraxcenter, Rotterdam, The Netherlands.

During a two year period 130 self-expanding wallstents (Medinvent, Lausanne) were implanted in 65 patients during 77 procedures in 91 coronary artery bypass graft stenosis. All patients had severe symptoms and were considered to be a high risk group: in 42 patients surgeons were reluctant or had refused to perform a new bypass operation, because of unfavourable coronary vessel anatomy. PTCA was not an attractive option in 47 patients because of the age of the grafts (mean 83 months), the length of the stenosis (mean 16.5 mm) and the unfavourable angiographic picture (tandem lesions (n = 21), lesions containing ulcers, aneurysm, calcifications or dissections (n = 34)). The mean unconstrained diameter of the stent was 4.3 (3.5 - 6.0 mm), implanted in grafts with a mean diameter of 3.3 (1.6 - 7.0 mm). The mean minimal luminal diameter of the stenosis was 1.4 ± 0.8 mm.
All procedures were technically successful and resulted in a significant increase of the mean minimal luminal diameter to 2.7 ± 0.7 mm. During hospital stay an acute thrombotic occlusion occurred in 7 patients (11%), necessitating reintervention in 4 patients (re-CABG) and leading to an acute myocardial infarction in 3 patients. Two patients died due to an intracerebral bleeding, directly related to the anticoagulation therapy given.
At late follow-up (3 - 6 months, n = 46), 16 patients (35%) developed a restenosis (> 50% DS) within the stent, necessitating reintervention in 15 patients (PTCA: n = 11; reCABG: n = 4). In the patient group without stent related restenosis (n = 30), 15 patients developed progression of native or bypass disease leading to recurrence of major angina pectoris symptoms, within 1 to 24 months. Ten of these patients underwent further intervention (Stent: n = 6; PTCA: n = 3; reCABG: n = 1).
Stenting in saphenous coronary bypass grafts is technically feasible and offers a new possibility in the difficult struggle to manage these patients with end stage coronary artery disease. The acute thrombotic occlusions and bleeding tendency associated with meticulous anticoagulant treatment cause anxiety but might be decreased by better patient selection and using newer designed stents. The restenosis rate (35%) seems to be lower than after conventional PTCA than in venous graft stenosis.

Wednesday, March 6, 1991
2:00PM–3:30PM, Room 216, East Concourse
Altered Endothelial Function in Heart Failure

2:00

PLASMA ENDOTHELIN CONCENTRATIONS ARE INCREASED IN HUMANS WITH CONGESTIVE HEART FAILURE

Richard J. Rodeheffer, Denise M. Heublein, Linda McKinley, John C. Burnett, Jr. Mayo Clinic, Rochester, MN.

Congestive heart failure (CHF) is characterized by decreased cardiac output and increased peripheral vascular resistance. CHF is known to be associated with increased sympathetic tone and by increases in plasma norepinephrine, renin, and arginine vasopressin. The degree of activation of these vasoconstrictor stimuli correlates poorly with the degree of heart failure. Hence, there is continued interest in identifying vasoconstrictors which may contribute to the pathophysiology of CHF. Endothelin (ET) is a peptide produced by endothelial cells which has potent and sustained vasoconstrictor effects. We sought to determine if plasma ET concentrations are increased in humans with symptomatic CHF. ET was measured by a newly developed sensitive radioimmunoassay to ET-1 (Amersham, UK). Plasma was extracted with C-8 Bond Elut cartridges with a recovery of 86%. Intra- and interassay variabilities are 6% and 9% respectively. Plasma ET concentrations were determined in a control group of 71 normal subjects and in 20 patients hospitalized for CHF (NYHA Class III-IV). The left ventricular ejection fraction in CHF patients was 24.1 ± 2.1%. Plasma ET in controls was 7.1 ± 0.1 pg/ml and in CHF patients 11.5 ± 0.9 pg/ml (p<0.001). We conclude that CHF in man is characterized by elevated plasma ET concentrations and that ET may contribute to the increased vascular resistance of CHF.

2:15

THE ROLE OF ENDOTHELIUM-DERIVED RELAXING FACTOR IN EXPERIMENTAL CONGESTIVE HEART FAILURE.

Fredric L. Hildebrand, Jr., Mark A. Perrella, and John C. Burnett, Jr.; Mayo Clinic and Foundation, Rochester, MN.

Congestive heart failure (CHF) is a state of marked vasoconstriction. While endothelium-derived relaxing factor (EDRF) is a potent vasodilator which mediates endothelium-dependent vascular relaxations in vitro, its role in the regulation of the peripheral circulation in CHF is unclear. We tested the hypothesis that inhibition of EDRF formation in CHF would fail to increase regional vascular resistances in experimental CHF, consistent with a depletion of EDRF in CHF. N-monomethyl-L-arginine (L-NMMA), a specific inhibitor of EDRF formation from L-arginine, was infused (50 μg/kg/min, i.v., for 70 minutes) into dogs with or without experimental CHF produced by rapid ventricular pacing for 8 days (Group I, n=7).

	Baseline	L-NMMA	Recovery
MAP (mmHg)	100±6	102±8	102±9
CO (L/min)	2.01±0.25	1.37±0.17#	1.25±0.15#
SVR (mmHg/L/min)	52.4±7.6	100.4±31.9#	65.9±9.7#
PVR (mmHg/L/min)	3.8±0.7	8.4±2.3#	7.8±1.0#
RVR (mmHg/ml/min)	0.61±0.08	0.94±0.13#	1.17±0.20#

(#p<0.05 vs. Baseline, MAP: mean arterial pressure, CO: cardiac output, SVR: systemic vascular resistance, PVR: pulmonary vascular resistance, RVR: renal vascular resistance)

L-NMMA administration in CHF resulted in marked increases in systemic, pulmonary, and renal vascular resistances. Moreover, when compared to the normal control group (Group II, n=5) receiving L-NMMA, these increases in regional vascular resistances were marked enhanced not attenuated. We conclude that experimental CHF is not characterized by an absence, but a functional presence of EDRF activity. These studies support an important functional role for EDRF as an antagonist to endogenous vasoconstrictors in the regulation of vascular tone in experimental CHF.